Wild Honey Poisoning: A Case Report

Olita Shilpakar,1 Bibek Rajbhandari,2 Bipin Karki3

1Department of General Practice and Emergency Medicine, T.U. Teaching Hospital, Kathmandu, Nepal,
2Department of General Practice and Emergency Medicine, Nepal Police Hospital, Kathmandu, Nepal,
3Department of Critical Care Medicine, Om Hospital and Research Centre.

ABSTRACT

Wild honey is consumed in many parts of the world as an alternative source of medicine with the belief of reducing cardiovascular, gastrointestinal and many other ailments. However, intoxication secondary to consumption of wild honey produced from the nectar of a few species rhododendrons has been encountered due to a toxin known as grayanotoxin. It is a sodium channel toxin causing symptoms like bradycardia, arrhythmias, hypotension, sweating, dizziness and altered sensorium. We report a case of a 58 year old man who presented to the emergency room following ingestion of wild honey with bradycardia, hypotension and altered mental status.

Keywords: bradycardia; grayanotoxin; hypotension; wild honey poisoning.

INTRODUCTION

Wild honey poisoning is caused by the ingestion of honey containing a toxin known as grayanotoxin. It is also known as rhodotoxin derived from the nectar of a few rhododendron species mostly found in the East black region of Turkey, Japan, Brazil, Spain and mountainous regions of Bhutan, India and Nepal.1 In Nepal, the consumption of wild honey has been considered as an alternative medicine serving medicinal as well as hallucinogenic purposes.2,3 Grayanotoxin binds to the receptors in the voltage gated sodium channels and manifests with parasympathetic overactivity like bradycardia, hypotension, dizziness and conduction defects.2,3 This case is an interesting description of the symptomatic presentation of a male patient following ingestion of wild honey and its management in the Emergency room.

CASE REPORT

A 58 year old male presented to the Emergency room of Tribhuwan University Teaching Hospital with the chief complaint of sudden onset of dizziness, sweating and nausea followed by altered sensorium in the form of drowsiness after ingestion of two tea spoonfuls of wild honey for dinner around 3 hours back. There was no history of chest pain, vomiting, pain abdomen, abnormal body movements or loss of consciousness in the patient. Patient was a known case of hypertension since the past 15 years and was under amlodipine 5mg tablets since then. He was also being treated with warfarin 4mg daily since the past three years for deep vein thrombosis of the right lower limb.

On examination, the patient was ill looking with a Glasgow coma scale score of 12/15 (E3V4M5). His radial pulse was not palpable and his heart rate was recorded by the monitor as 37 beats per minute, his blood pressure was 80/60mm Hg, his respiratory rate was 20 breaths /minute, he was afebrile with a temperature of 97 degrees F and he was maintaining 86% saturation in room air. His chest auscultation revealed bilateral decreased air entry, however, his neurological and gastrointestinal examination revealed no other abnormalities.
A 12 lead electrocardiogram revealed extreme bradycardia with a heart rate of 37 beats per minute (Figure 1). His routine hematological and biochemical investigations including cardiac enzymes and an arterial blood gas analysis were within normal limits. An echocardiogram revealed mild mitral regurgitation and mild tricuspid regurgitation with a LVEF of 60%. The patient was provided oxygen supplementation at 2 litres per minute via nasal prongs and was managed by administering an intravenous dose of atropine 0.6mg and 500ml of 0.9% normal saline. One more dose of 0.6mg atropine was administered via intravenous route after 5 minutes since the patient was still bradycardic with a heart rate of 40 beats per minute, following which his heart rate increased to 93 beats per minute with a normal sinus rhythm in the 12 lead electrocardiogram. (Figure 2). His blood pressure was 110/70mmHg, his Glasgow coma score improved to 15/15 and his symptoms gradually resolved over the next 2 hours. He was kept under observation with strict hemodynamic monitoring over the next 24 hours which was uneventful. He was finally discharged from the hospital under his regular medications.

**Figure 1. ECG on admission, heart rate: 37 beats/minute**

**Figure 2. ECG after recovery, heart rate 93 beats/minute**

**DISCUSSION**

Grayanotoxin, also known as andromedotoxin or rhodotoxin, is derived from the leaves or flowers of plants belonging to genera of the Ericaceae family comprising the Rhododendron, Agarista and Kalmia genera and is found growing in the hilly areas of Turkey, Brazil, Japan and parts of North America, as well as in India, Bhutan and Nepal. 1, 2 Out of the 18 forms of grayanotoxins identified till date, Grayanotoxin 1 and 2 have been found in the honey, leaves and flowers of Rhododendron ponticum and Rhododendron flavum which are considered as the most potent forms of the toxin. 2, 3

Grayanotoxin binds to sodium channels in cell membranes, thus increasing their permeability, inhibiting repolarisation and maintaining a state of depolarisation. The action potential is weakened by the increasing inward current and decreasing outward current of sodium on the cell membrane at the sinoatrial node resulting in sinus node dysfunction. The stimulation of the unmyelinated afferent cardiac branches of the vagus nerve causes tonic inhibition of central vasomotor centres with a reduced sympathetic output and reduced peripheral vascular resistance leading to cardiovascular symptoms. 4

Gastrointestinal and neurological symptoms have also been encountered rarely, however; cardiovascular side effects of grayanotoxin are more common and include hypotension, bradycardia, complete
atrioventricular block and myocardial infarction. There have been case reports from Turkey and Nepal in which the patients responded to intravenous fluids and IV atropine administration which was similar to our case report.\textsuperscript{3,6} Onat et al also showed that the non-selective muscarinic antagonist atropine sulphate alleviated bradycardia, while the selective M2-muscarinic antagonist AF-DX 116 restored heart rate.\textsuperscript{5} The toxic effects usually last for 24 hours recovering after symptomatic management and is rarely fatal in humans.

Honey consumption is common in our part of the world since it is believed to have a wide range of health benefits, however, the precise therapeutic dose is not yet documented, so more studies on the therapeutic and toxic doses of honey and its life threatening side effects are required. Immediate symptomatic management and close observation is the key to the management of such patients. Determination of grayanotoxins in leaves, flowers and extracts via spectrography technique could be considered in the near future.

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CONFLICT OF INTEREST: None.

REFERENCES


